The average loss of total nitrogen by urine and stool in these five cases was 12.9 gm. per day. The average loss in nitrogen from metabolized proteid as shown by the total nitrogen of urine was 11.7 gm. per day.

The largest amount of nitrogen we were able to supply daily by rectal feeding was 3.9 gm., of which 2.06 gm. were returned with stools. According to the observations of Fr. Müller, the stools of individuals taking absolutely no nourishment contains 0.2 gm. per day. With this deduction from our estimations of the return nitrogen we have in no case observed an absorption of more than 50 per cent. of the proteid supplied, and it has fallen as low as 30 per cent. The average amount of nitrogen absorbed in these cases was 1.14 gm. per day.

When we compare the amount of nitrogen lost by these cases with the amount it was possible for them to absorb by colonic feeding, we realize how little we accomplish.

The difference between nutritional enteroclysis and normal salt enteroclysis was a little over one-tenth of the tissue albumin loss per day.

Over the periods of time that rectal feeding is usually carried out it is from practical standpoints an almost negligible quantity.

INTRACRANIAL TELANGIECTASIS: SYMPTOMATOLOGY AND TREATMENT, WITH REPORT OF TWO CASES.[1]

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Operations for Jacksonian epilepsy have so frequently resulted in negative findings that I desire to call attention to a group of cases which yields to treatment. This group, though apparently not large, has, nevertheless, a characteristic pathological picture. I shall briefly consider this condition from three points of view: (1) symptomatology; (2) pathology; (3) treatment. This paper is based on two typical cases.

Case I (Private record).—R. D., a boy, aged ten years, whom I saw in 1909, had, three months after a fall, fever and convulsions confined to the left side of the body, which began in the leg. A complete left-sided paralysis developed, which cleared up in a month. Following this the patient was apparently well and bright at school during the next year; then he began to have monthly convulsions, beginning in the face, with marked irritability; occasional headaches and vomiting; he often started suddenly. Wasser-

[1] Read at the meeting of the American Neurological Association, New York, May 6 to 8, 1915.

mann negative. The physical examination revealed a left knee-jerk more active than the right; a double Babinski and normal eye-grounds; color fields could not be taken on account of the youth of the patient. On opening the dura (Fig. 1) a huge bluish mass of vessels protruded, which were so dense in places that the underlying cortex could not be seen. This mass lay over the motor area. There were no branches connecting with the dural vessels. I considered it inadvisable to try to remove this mass. Following the operation the patient had acute suppression of urine for twenty-

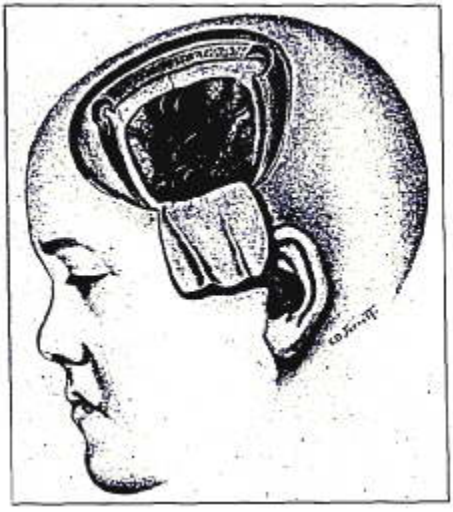

FIG. 1.—R. D., aged ten years. The very large size of the vessels of this telangiectasis is not sufficiently indicated on this drawing.

four hours, and then made an uneventful recovery. His condition remained unimproved. Therefore, at the family's urgent request, a month afterward I tried to remove this angiomatous mass, but the patient died twenty-four hours later. Autopsy showed grossly that the process was confined to the cortex. Unfortunately, no microscopic examination was made.

CASE II.—C. E., a boy, aged ten years, surgical number 1873. Admitted February 8, 1915, discharged February 23, 1915. First seen in November, 1914. Referred by Dr. Schwab and Dr. Wolfner.

His past history was unimportant. First convulsion when three months old. There have been five since, three of these in the past three months. Unconsciousness lasted from one to two hours. All convulsions begin in the wrist of the left hand. There are minor attacks, occurring as often as five or six times a day. In these the patient seems sleepy and has a numb feeling in his hand. Wassermann negative. Physical examination showed a weakness of the two lower branches of the left seventh nerve on emotional exertion, and a pale telangiectasis over the forehead. The left eye-ground

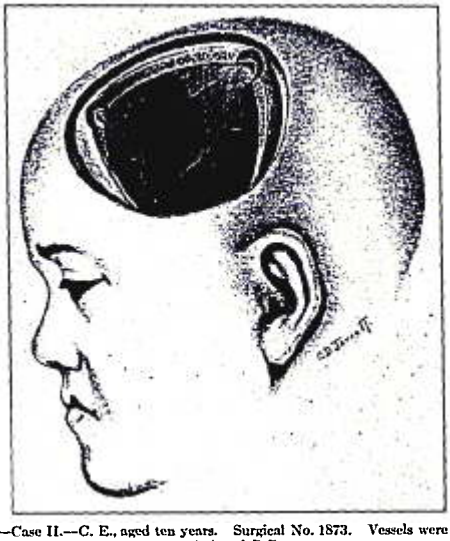

FIG. 2.—Case II.—C. E., aged ten years. Surgical No. 1873. Vessels were ligated at *A A* and *B B*.

showed an old choroidal atrophy; the right eye was normal. Again, the color fields could not be satisfactorily taken on account of the youth of the patient. An exploratory craniotomy was performed in February, 1915. An angiomatous process was found in the dura, which on reflecting the dura was found to have numerous connections with the pial vessels (Figs. 2 and 3). This area of increased vascularity lay over the lower part of the motor area. All the vessels connecting with the pial vessels were ligated and the dural mass ligated above and below; then the dura was closed. Two days after

the operation the patient had a violent convulsion. Since then he has been well. He has had three of the light attacks, of which he formerly had five to six daily. It is too early to express an opinion as to the ultimate result.

Both of these cases showed an unusual vascularity: the one of the vessels of the dura, which connected with the pial vessels, the other of the superficial vessels of the cortex.

In a careful survey of the literature I find that there really are very few cases like my second case. Virchow cites a case described

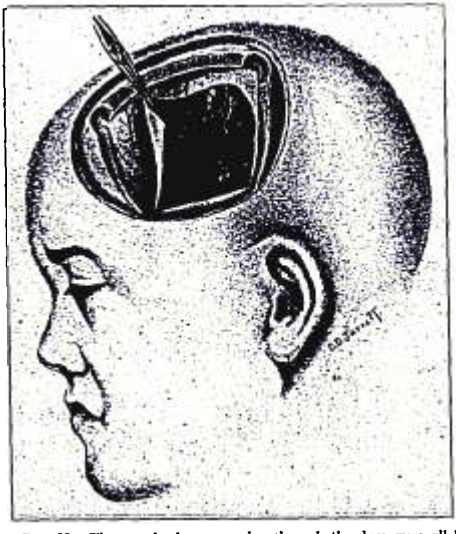

FIG. 3.—Case II.—The vessels shown running through the dura were all ligated.

by Hooper in his *Morbid Anatomy of the Human Brain*, published in 1828, which corresponds fairly closely. Pean gave a brief account before the French Academy of Medicine of an "angiome intracranien des meninges," which seems to have been identical.

Dr. Cushing, in 1906, described three cases of intracranial hemorrhage associated with trigeminal nevi. The process in all three of his cases was in the dura. He does not, however, mention the possibility that an identical process might occur on the cortex.

Oppenheim, in January, 1913, reported several similar cases in his paper on congenital brain tumors. He has given an admirable clinical picture of a condition which he calls angioma, which, in my opinion, is not an angioma at all but an entirely different pathological entity.

The first case is a type of which more have been observed. Krause in his book on the *Surgery of the Brain and Spinal Cord* reports two cases, one of which he has illustrated. This he calls an angioma venosum racemosum. In his opinion, evidently, he is dealing with a new growth—a true tumor. Tooth in his series of five hundred cases from the National Hospital has not a single instance of either condition. Bruns in his book on *Brain Tumors* makes the statement that cavernous angiomata and telangiectatic tumors have been found fairly frequently at operation,[2] and reports one case of what he calls an angioma which he observed himself. As he gives insufficient data it is impossible to decide to which of these two groups this case belonged. Virchow studied and described a number of such conditions, and was the first to clearly differentiate between telangiectases and true angiomata. He drew attention to the point which Adami emphasizes, namely, that a telangiectasis is a congenital dilatation of capillaries without any new-formed bloodvessels, while an angioma is a new growth and only properly so called when new bloodvessels are formed. In some of these cases the process is connected primarily with the capillaries, in some with the veins, and in others with the arteries. In true angiomata no vessels with all three coats developed are found, but in both of my cases the vessels looked prefectly normal, and were well formed.

In looking through the literature I find that these points are lost sight of and that the term angioma racemosum arteriale, or venosum, has been applied to conditions which were not actually new growths. I question very much whether Krause's case is correctly named. This, by the way, is the case cited by Oppenheim. It is the counterpart of my first case, and impresses me more like a telangiectatic process. Another point of interest is that cases like the two I have here reported are frequently, possibly always, associated with telangiectases elsewhere on the body, particularly on the face. My second case showed this. This was observed in Pean's case, also in Dr. Cushing's cases. Such cases as that of Emanuel, which he made the basis of an exhaustive discussion of angioma racemosus, are undoubtedly true new growths.

An etiological factor that has often been mentioned is trauma. Exactly what the relation of intracranial growths and trauma is, if any, is not at all clear. In certain cases it seems highly probable

[2] At the meeting at which this paper was read, several members reported on published cases.

that a trauma has either irritated a preëxisting new growth, making it develop rapidly, or has actually been the cause of the new growth. In my first case there was a definite history of this sort, but the operative findings, it seems to me, exclude that factor, since there was no evidence of inflammatory reaction such as adhesions, connective-tissue overgrowth, or roughened inner layer of the dura overlying the dilated vessels.

The classification of this process might seem merely of academic interest, but it has most important bearing on the treatment and prognosis. If this is a true new growth, extirpation is the procedure indicated, while if it is a telangiectasis, ligation should suffice. The question naturally arises, Will these thrombosed vessels, if left *in situ*, irritate the cortex, and should they therefore be removed after ligation? If possible the ligation should be planned so that the vessels do not remain filled with blood but are collapsed after the ligation. They will then atrophy and do no harm. This condition is a favorable one from an operative and prognostic stand-point.

It seems to me essential to distinguish more carefully between a true angioma—a neoplasm—and the process observed in these two cases, which is congenital and not a neoplasm. Undoubtedly some of the cases called angiomata have been misnamed. These cases, when reclassified, constitute a group which must be kept in mind in every case of Jacksonian epilepsy.

The clinical picture is quite a definite one.

1. Attacks of Jacksonian epilepsy occurring at long intervals in non-syphilitic young persons.
2. Unconsciousness of long duration.
3. No evidence of increased intracranial pressure.
4. A very slow progression of symptoms.
5. Telangiectases on the head or face.

For this condition I should like to suggest the name of intracranial telangiectasis.

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RESPIRATORY SIGNS AND SYMPTOMS IN TRICHINOSIS.

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One hundred and two case histories of trichinosis have been reviewed to determine the frequency of associated respiratory signs and symptoms which Dr. Edsall suggested may be more common